

Evaluating and Managing Asymptomatic AV Block in young : When to Intervene?

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ABSTRACT

High-degree atrioventricular (AV) block represents a serious cardiac conduction disorder characterized by the complete interruption of electrical signals between the atria and ventricles. This condition, which can manifest at various levels of the conduction system, poses a significant risk of mortality due to complications such as heart failure and sudden cardiac death if left untreated. This article presents a case study of a 46-year-old asymptomatic female patient with a high-degree AV block, who exhibited no significant clinical or etiological findings after extensive evaluation.

The management of asymptomatic AV block remains a complex decision-making process, balancing the potential benefits of medical versus interventional treatment, including the implantation of a permanent pacemaker. Current guidelines recommend a thorough etiological assessment to identify any reversible causes of AV block before proceeding with permanent pacing, particularly in asymptomatic patients.

Additionally, for AV blocks of unknown etiology, the decision to implant a pacemaker should be carefully considered, taking into account all associated risks and potential long-term outcomes.

This review discusses the clinical considerations, diagnostic evaluations, and management strategies for highdegree AV block, with a focus on asymptomatic cases, and highlights the need for individualized patient care based on the underlying etiology and clinical presentation.

Keywords: Uncertain causes; Non-reversible; Pacemaker; Asymptomatic; Young adult,; Atrioventricular block

INTRODUCTION

High-degree atrioventricular block (AV block) is a critical intracardiac conduction disorder that is defined by the complete and persistent interruption of electrical conduction between the atria and the ventricles. This blockade can occur at various levels, including the atrioventricular node (nodal block), the bundle of His (Hissian block), or even further down the conduction system (infra-Hissian block).

In the absence of appropriate medical intervention, patients with this condition are at a significantly elevated risk of mortality due to heart failure, which results from reduced cardiac output, or sudden cardiac death, which may occur as a consequence of asystole or prolonged bradycardia that precipitates life-threatening ventricular tachyarrhythmias.

CLINICAL OBSERVATION

We present the case of Mrs. Z.Y., a 46-year-old woman with a notable family history of cardiovascular disease and thyroid disorders. Her medical history is significant for hypertension, which is being managed with angiotensin-converting enzyme (ACE) inhibitors, and she has been under surveillance for multinodular thyroiditis for the past year. Additionally, she underwent cholecystectomy 15 years ago, and two of her sisters have been diagnosed with Hashimoto's thyroiditis. It is important to note that the patient denies taking any regular medications outside of her prescribed antihypertensive therapy.

Upon clinical evaluation, the patient was found to be asymptomatic. Her vital signs revealed a blood pressure of 130/65 mmHg and a bradycardic heart rate of 45 beats per minute. Physical examination, including cardiac auscultation, was unremarkable, and there were no signs suggestive of heart failure or other systemic abnormalities.



Figure 1 : The electrocardiogram reveals the presence of a complete bundle branch block (BAV).



Na+: 141mmol/l	K+: 4,1mmol/l	Mg2+ : 0,84mmol /1	C a2+ : 96,6mg/l
ALAT : 20U/1	ASAT : 22U/l	GGT : 47U/l	PAL: 92U/1
Urée : 0,29g/l	Creat: 7,34mg/l	DFG : 89,2	
TSH : 2,48µUI/ml	T4 : 14,3pmol/l	T3:4,4pmol/l	
GB:8 020	Lymph: 2 730	HBG: 13,2g/dl	Pq: 236 000
Sérologies : TPHA- VDRL : négatives HVC –HVB : négative HIV : Négative De Lyme : Négative		BGSA : Sans and Enz de conversion	malies : N

Given her clinical presentation, a series of diagnostic evaluations were performed to assess the extent and cause of the AV block. These included a 24-hour Holter monitor to verify the permanence of the AV block, a stress test to rule out ischemic heart disease, and extensive imaging studies, including thoracic echocardiography, coronary angiography, and chest computed tomography (CT). All investigations returned without significant findings, leaving the patient diagnosed with an asymptomatic high-degree AV block with a negative etiological workup.



Figure 2: The Holter ECG recording reveals a complete bundle branch block (BAV

DISCUSSION

Approach to Asymptomatic Atrioventricular Block

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In asymptomatic patients diagnosed with high-degree AV block, clinicians face a complex decision regarding the best approach to management. The options generally revolve around either continuing medical management or proceeding with interventional strategies such as the implantation of a permanent pacemaker (PM).

Medical Management:

According to the 2018 guidelines released by the American College of Cardiology/American Heart Association/Heart Rhythm Society (ACC/AHA/HRS), for patients with second or third-degree AV block who present with symptoms and are hemodynamically unstable, initial treatment with atropine is recommended to enhance heart rate and alleviate symptoms. In cases where an ischemic cause is unlikely, beta-agonists such as isoproterenol, dopamine, dobutamine, or epinephrine may be considered, although the supporting evidence for these interventions remains limited.^[1]

Pacemaker Implantation:

The 2013 guidelines from the European Society of Cardiology (ESC) provide clear guidance regarding the use of pacemakers in patients with AV block. They advise against the implantation of a permanent pacemaker in cases where the AV block is attributable to a reversible cause.^[2] This approach is echoed by the American guidelines, which similarly do not recommend pacemaker implantation for AV blocks that are due to reversible and non-recurrent conditions.^[1]

Etiological Assessment:

Identifying the underlying cause of AV block is paramount in guiding treatment. The 2018 American guidelines stress the importance of a thorough etiological assessment, as the causes of AV block are diverse and range from congenital defects to acquired conditions.^[1]

Congenital Causes: Congenital AV block is typically diagnosed at birth or via antenatal screening. It can occur as an isolated defect or in conjunction with other congenital heart abnormalities. Additionally, congenital AV block may be associated with maternal autoimmune conditions, such as systemic lupus erythematosus or Sjögren's syndrome, where maternal antibodies cross the placenta and affect the fetal heart.

Ischemic Heart Disease: Acute coronary syndromes, coronary artery disease, and other ischemic conditions are significant contributors to the development of AV block, particularly in the context of myocardial infarction affecting the conduction system.

Infectious Causes: Infective endocarditis, particularly when complicated by valvular abscesses, can lead to AV block due to the involvement of the conduction system. Other infectious causes include Lyme disease, especially in endemic areas, and rheumatic fever. A meta-analysis of AV block in Lyme disease cases demonstrated that the duration of conduction abnormalities could vary widely, with some cases resolving in days and others persisting for weeks or months.^[3]



Inflammatory and Infiltrative Diseases: Conditions such as sarcoidosis, cardiac amyloidosis, and other connective tissue disorders can infiltrate the heart's conduction system, leading to AV block. Sarcoidosis, in particular, is an important differential in younger patients presenting with AV block.

Metabolic Disorders: Thyroid dysfunction, particularly hypothyroidism, electrolyte imbalances, and acid-base disturbances can also precipitate AV block.

Degenerative Causes: In older adults, degenerative changes in the conduction system, often due to fibrosis, are a common cause of AV block.

A case report describes a patient with a suspected sleep apnea syndrome, where polysomnography detected a 6-second pause coinciding with apnea, occurring prior to hypoxia. This finding highlights the importance of conducting Holter-ECGs and considering the diagnosis of nocturnal pauses.^[4]

Iatrogenic causes, particularly those related to medications, require careful assessment, especially in the context of procedures such as ablation of accessory septal pathways, valve replacements (notably aortic valve replacement), transcatheter aortic valve implantation (TAVI), or septal alcohol ablation for hypertrophic cardiomyopathy (HCM).

Category	Conditions
Congenital/genetic	Congenital AV block (associated with maternal systemic lupus erythematosus), Congenital heart defects (e.g., L-TGA), Genetic (e.g., SCN5A mutations)
Infectious	Lyme carditis, Bacterial endocarditis with perivalvar abscess, Acute rheumatic fever, Chagas disease, Toxoplasmosis
Inflammatory/infiltrative	Myocarditis, Amyloidosis, Cardiac sarcoidosis, Rheumatologic diseases: Systemic sclerosis, SLE, RA, reactive arthritis (Reiter's syndrome), Other cardiomyopathy—idiopathic, valvular
Ischemic	Acute MI, Coronary ischemia without infarction—unstable angina, variant angina, Chronic ischemic cardiomyopathy
Degenerative	Lev's and Lenegre's diseases

Table 1: Etiological Factors Contributing to Atrioventricular Block (AV Block).



Vagotonic/Associated with	Vagotonic-associated with increased vagal tone		
Increased Vagal Tone	Sleep, obstructive sleep apnea		
	High-level athletic conditioning		
	Neurocardiogenic		
Metabolic/Endocrine	Acid-base disorders		
	Poisoning/overdose (e.g., mercury, cyanide, carbon monoxide, mad honey)		
	Thyroid disease (both hypothyroidism and hyperthyroidism)		
	Adrenal disease (e.g., pheochromocytoma, hypoadosteronism)		
Other Diseases	Neuromuscular diseases (e.g., myotonic dystrophy, Kearns-Sayre syndrome, Erb's dystrophy)		
	Lymphoma		
latrogenic	Medication-related		
	Beta blockers, verapamil, diltiazem, digoxin		
	Antiarrhythmic drugs		
	Nutraceuticals		
	Catheter ablation		
	Cardiac surgery, especially valve surgery TAVR, alcohol septal ablation		

Numerous drugs with established causal links include non-cardioselective beta-blockers (BBs) used for glaucoma treatment. Literature reports reversible causes, exemplified by a case of complete atrioventricular block (AVB) associated with timolol eye drops due to systemic absorption.^[5] Central antihypertensives, bariatric ion channels (ICs), class I and III antiarrhythmics, antipsychotics, antidepressants, as well as anesthetics, digoxin, and cannabinoids, have also been implicated.

Table 2: Medications That Can Induce/Exacerbate Bradycardia or Conduction Disorders

Antihypertensive	Antiarrhythmic	Psychoactive	Others



Beta adrenergic	Adénosine	Donepezil	Anesthetic drugs
receptor blockers	Amiodarone	Lithium	/propofol
(including beta	dronedarone	Opioid analgesic	Cannabis
adrenergic blocking eye	Flecainide	Phenothiazine	Digoxine
drops used for	Procainamide	antiemetics and	Ivabradine
glaucoma)	Propafenone	antipsychotics	Muscle relaxant
Clonidine	Quinidine	Phenytoin	/succinylcholine .
Methyldopa	Sotalol	Selective serotonin	
Non-dihydropyridine		reuptake inhibitors	
calcium channel		Tricyclic	
blockers		antidepressants	
Reserpine			

What assessment should be conducted ?

American guidelines recommend the following assessments for patients with a conductive disorder:^[1]

Biological Assessment: For all patients, a biological evaluation should include thyroid function tests, Lyme serology, and measurements of potassium and bicarbonates to investigate potential acidosis based on clinical indications.

Imaging for 2nd or 3rd Degree Mobile BAV: In cases of second or third-degree mobile atrioventricular block (AVB), transthoracic echocardiography (ETT) is advised. Additional imaging with transesophageal echocardiography (TEE), cardiac MRI, and scintigraphy may be considered if structural heart disease is suspected.

Imaging for Asymptomatic 1st Degree BAV: For patients with asymptomatic first-degree AVB or in the absence of suspected structural heart disease, routine imaging is not recommended.

<u>Assessment for Nocturnal Conductive Disorders:</u> In patients with nocturnal conductive disorders, polysomnography should be performed to evaluate for sleep apnea syndrome (SAS). Patients with a documented conductive disorder associated with SAS should be treated with continuous positive airway pressure (CPAP) at night. Weight loss should also be considered.

<u>Genetic Counseling</u>: For patients with a conductive disorder linked to a genetic mutation, genetic counseling is recommended.

What course of action should be taken in the case of a bundle branch block (BAV) of unknown etiology?

A Danish study published by the European Society of Cardiology in 2019 analyzed patients with bundle branch block (BAV) who were fitted before the age of 50 over a 20-year period (1996-2015). The study found that 50%



of the cases had an unknown etiology, followed by iatrogenic causes, with ischemic and infectious causes being less common. In a subsequent study from the same Danish registry, researchers followed patients with BAV of unknown etiology who were fitted before age 50 for 20 years. They compared these patients with a matched normal population, evaluating all-cause mortality, hospitalization for congestive heart failure (CHF), cardiovascular mortality, and hospitalization for ventricular tachycardia (VT). The findings indicated that the risk of these complications was significantly higher in the BAV cohort, with a threefold increase in all-cause mortality, a tenfold increase in hospitalization for CHF, a twelvefold increase in cardiovascular mortality, and a fourteenfold increase in VT occurrence, as well as nearly a fivefold increase in the composite criterion.^[6]

American guidelines recommend treating the underlying cause in patients with BAV of a reversible origin.^[1] For patients on a stable dose of beta-blockers (BBs) or other antiarrhythmics, it is reasonable to fit a pacemaker without waiting for treatment reversibility.^[1] In cases of BAV associated with cardiac sarcoidosis, fitting a defibrillator is advisable. A study published by the American Heart Association (AHA) in 2011 showed that BAV due to sarcoidosis results in more frequent ventricular fibrillation (VF), VT, and cardiac mortality compared to idiopathic BAV, emphasizing the benefit of a defibrillator.^[7]

For patients with BAV related to dysthyroidism (excluding myxedema), a device can be recommended without awaiting reversibility under treatment. This recommendation is supported by a Turkish study, which found that the reversibility of the block following correction of the thyroid disorder is highly variable among individuals.^[8]

CONCLUSION:

In managing high-degree AV block, particularly in asymptomatic patients, the decision-making process should be guided by a comprehensive assessment of the underlying etiology. In cases where a reversible cause is identified, addressing the underlying condition should be the primary focus. However, for patients with AV block due to non-reversible or uncertain causes, and in those with even asymptomatic, the consideration of permanent pacing may be warranted to prevent potentially fatal complications. The management strategy should be individualized, balancing the risks and benefits of intervention based on the patient's clinical profile and the underlying cause of the conduction disorder.

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